ABSTRACT

Pemphigus vulgaris (PV) is an autoimmune disease with a possible fatality of the skin and mucosae which is induced by an antibody against desmoglein 3 (Dsg3). Persistent production of anti-Dsg3 IgG can be induced by adoptively transferring spleen cells of a DSG3-/- mouse immunized with rDsg3 into an RAG2-/- immunodeficient mouse expressing Dsg3 protein. This IgG in the blood binds to the Dsg3 protein *in vivo*, induces the breakage of intercellular adhesion of keratinocytes and thus brings about the phenotype of pemphigus vulgaris involving the formation of blisters in the oral mucosa and the disappearance of resting hair. These effects are sustained over 6 months. By using this method, active disease model animals relating to various autoimmune diseases can be constructed.